

The effect of hypoxia on performance during 30 s or 45 s of supramaximal exercise

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Summary. The purpose of this study was to evaluate the effect of hypoxia ($10.8 \pm 0.6\%$ oxygen) on performance of 30 s and 45 s of supramaximal dynamic exercise. Twelve males were randomly allocated to perform either a 30 s or 45 s Wingate test (WT) on two occasions (hypoxia and room air) with a minimum of 1 week between tests. After a 5-min warm-up at 120 W subjects breathed the appropriate gas mixture from a wet spirometer during a 5-min rest period. Resting blood oxygen saturation was monitored with an ear oximeter and averaged $97.8 \pm 1.5\%$ and $83.2 \pm 1.9\%$ for the air (normoxic) and hypoxic conditions, respectively, immediately prior to the WT. Following all WT trials, subjects breathed room air for a 10-min passive recovery period. Muscle biopsies from the vastus lateralis were taken prior to and immediately following WT. Arterialized blood samples, for lactate and blood gases, were taken before and after both the warm-up and the performance of WT, and throughout the recovery period. Open-circuit spirometry was used to calculate the total oxygen consumption ($\dot{V}_{O_2}$), carbon dioxide production and expired ventilation during WT. Hypoxia did not impair the performance of the 30-s or 45-s WT. $\dot{V}_{O_2}$ was reduced during the 45-s hypoxic WT (1.71 ± 0.21 l) compared with the normoxic trial (2.16 ± 0.26 l), but there was no change during the 30-s test (1.22 ± 0.11 vs 1.04 ± 0.17 l for the normoxic and hypoxic conditions, respectively). Muscle lactate (LA) increased more during hypoxia following both the 30-s and 45-s WT (67.1 ± 25.0 mmol·kg⁻¹ dry weight) compared with normoxia (30.8 ± 18.0 mmol·kg⁻¹ dry weight). Hypoxia did not influence the change in intramuscular adenosine triphosphate, creatine phosphate and glucose-6-phosphate. The performance of WT during hypoxia was associated with a greater decrease in muscle glycogen ($P < 0.06$). Throughout the recovery period, blood LA was lower following the hypoxia (8.43 ± 2.88 mmol·l⁻¹) compared with normoxia (9.15 ± 3.06 mmol·l⁻¹). Breathing the hypoxic gas mixture prior to

the performance of WT increased blood pH to 7.44 ± 0.03 , compared with 7.39 ± 0.03 for normoxia. Blood pH remained higher during the 10-min recovery period following the hypoxic WT trials (7.24 ± 0.08) compared with the normoxic WT (7.22 ± 0.06). Blood P_{CO_2} was reduced prior to and immediately following WT during hypoxia, but there were no differences between the normoxic and hypoxic trials during the 10-min recovery period. These data indicate that more energy was transduced from the catabolism of glycogen to lactate during the hypoxic WT trials, which offset the reduced O_2 availability and maintained performance comparable with normoxic conditions. It is suggested that the induced respiratory alkalosis associated with breathing the hypoxic gas could account for the increased rate of muscle LA accumulation.

Key words: Anaerobic power – Exercise – Metabolism – Lactate – Acid-base – Oxygen consumption – Skeletal muscle

Introduction

A maximal effort of cycling or running for 30 s results in a marked decrease in intramuscular adenosine triphosphate (ATP), creatine phosphate (CP) and glycogen, and a pronounced increase in glycolytic intermediates and lactate (LA) (Cheetham et al. 1986; Jacobs et al. 1982, 1983). Large increases in blood LA and decreases in blood pH have been reported also following a 30-s maximal exercise test (Cheetham et al. 1986; Inbar et al. 1983; Jacobs 1980; Macdonald et al. 1983; Taunton et al. 1981). These documented changes in both intramuscular and blood metabolites reflect the substantial energy contribution from anaerobic sources to the total ATP demand during maximal-effort, short-duration exercise.

In contrast, the relative importance of the energy contribution from aerobic sources to the total energy

demand is not well defined for exhaustive exercise of short duration. The energy equivalent of the total oxygen consumption ($\dot{V}_{O_2}$) measured during the 30-s Wingate test (WT) by open-circuit spirometry has been estimated to represent 15–20% of the total energy demand (Kavanagh and Jacobs 1988). This latter study, however, did not account for the fact that during the transition from rest to exercise, $\dot{V}_{O_2}$ measured at the mouth underestimates tissue $\dot{V}_{O_2}$ due to changes in tissue myoglobin and venous blood O_2 stores (Inman et al. 1987). Therefore, the relative contribution from aerobic energy sources could exceed 20% of the total energy demand for a 30-s supramaximal exercise test. In light of the frequent use of the WT as a test of anaerobic power, we thought it appropriate to clarify further the aerobic component. The present study was designed, therefore, to examine the influence of a reduced contribution from aerobic energy sources on the performance of short-duration supramaximal exercise.

It was hypothesized that if the performance of a 30-s WT was not affected by a decreased O_2 availability, then changes in intramuscular metabolites would reflect an enhanced contribution from anaerobic energy sources. Conversely, if the rate of ATP production from anaerobic metabolism was not increased when O_2 availability was reduced, then the performance of the 30-s maximal effort would be impaired. To potentiate the possible influence of a decreased contribution from aerobic energy sources on the performance of short-duration exhaustive exercise both a 30-s and 45-s WT were compared while subjects breathed either 21% (normoxia) or 10–12% O_2 (hypoxia).

Methods

Subjects. Following approval from the Human Ethics Committee, 12 male volunteers participated in the study after giving written informed consent. Mean values $\pm$ SD for age, mass, percent body fat, treadmill $\dot{V}_{O_{2\max}}$ and muscle fibre type in the vastus lateralis were 27.9 ± 3.4 years, 88.4 ± 18 kg, $12.4 \pm 3.5\%$, 52.2 ± 6.8 ml $\cdot$ kg $^{-1} \cdot$ min $^{-1}$ and $42.5 \pm 14.7\%$ slow twitch, respectively.

Test procedures. All testing was carried out at an altitude of 200 m above sea level. Subjects were randomly assigned to either a 30-s or 45-s WT condition. There were no differences between groups for the subjects' physical characteristics described above. The WT consisted of pedalling at maximal frequency on a mechanically braked cycle ergometer (Cardionics, Stockholm, Sweden) with the resistance set so one pedal revolution resulted in 4.4 J work $\cdot$ kg $^{-1}$ body weight. The number of revolutions performed during each 5-s interval was counted electronically and the power output (watts) calculated. The peak power output (PPO) was defined as the highest power output during a 5-s interval. The mean power output (MPO) represented the average over the entire 30-s or 45-s interval, and the fatigue index (FI) was the final 5-s power output expressed relative to PPO.

Each subject reported to the laboratory on 3 separate days. The 1st day consisted of a maximal treadmill test to determine $\dot{V}_{O_{2\max}}$ followed by a 30-min rest prior to performing a 30-s WT. The latter was a familiarization procedure to allow the subjects to become accustomed to the breathing apparatus and the WT. For the two remaining WT trials, which were performed a minimum of 1 week apart, the subjects reported to the laboratory at least 2 h

post prandial. The order of these two testing trials (i.e. hypoxia or normoxia) was balanced among subjects and was performed in a single-blind manner. Verbal encouragement was given to all subjects during the performance of the WT.

A resting muscle biopsy of the vastus lateralis was performed and a 21-gauge butterfly catheter was inserted into a superficial vein in the dorsum of the hand prior to each test. The catheter was kept patent by periodic flushing with heparinized saline (approximately 10 IU heparin $\cdot$ ml $^{-1}$). The subject then stood at the bicycle ergometer with his hand and forearm resting on a support table covered with a heating chamber that increased the skin temperature to 42 – 45° C. This temperature was maintained throughout each trial and was verified with a skin thermistor placed close to the site of blood sampling. The technique of heating the blood sampling area has been shown previously to produce a valid measure of arterial pH and P_{CO_2} (Forster et al. 1972). The P_{O_2} values were only used for verification of arterialization: at rest (prior to breathing a gas mixture) P_{O_2} exceeded 65–70 mmHg for all subjects.

The subjects were then given a warm-up of cycling for 5 min at 120 W, followed by a minimum of 5-min rest, during which they sat passively on the cycle and inspired the gas mixture from a 350-l and a 150-l wet spirometer, connected in series. The subjects breathed through a low-resistance Hans Rudolf valve with the inspired side connected to the spirometer, which contained either air (normoxia) or a 10–12% oxygen plus 88–90% nitrogen mixture (hypoxia). An ear oximeter (Biox II) was used to determine oxygen saturation just prior to WT performance. The oxygen saturation was required to be 85% or less during the hypoxic condition, before the performance of WT. Immediately upon completion of the WT, the mouth piece was removed and the subjects inspired room air throughout the recovery period. A muscle biopsy and blood sample were taken as soon as possible after exercise (within 10 s for the biopsy and 20 s for the blood sample) with the subject seated on the cycle. After these samples had been obtained, the subjects were transferred to a cot, where they lay passively during the recovery period.

During the performance of the WT, open circuit spirometry was used to determine total $\dot{V}_{O_2}$, carbon dioxide production ($\dot{V}_{CO_2}$) and expired ventilation ($\dot{V}_E$). The expired gases were directed into a 5-l mixing box and through an Alpha Technologies

Table 1. Inspired oxygen concentration, oxygen saturation and cardiorespiratory responses to the 30-s and 45-s Wingate test (WT) under normoxic and hypoxic conditions

	30-s		45-s	
	Normoxia	Hypoxia	Normoxia	Hypoxia
Inspired oxygen concentration (%)	20.9	10.7* (0.9)	20.9	10.7* 0.3
Oxygen saturation (%)	98.7 (0.5)	83.0* (2.7)	97.0 (1.8)	83.3* (0.8)
$\dot{V}_E$ (l)	50.2 (8.8)	46.1 (8.3)	84.5 (14.7)	90.4 (17.2)
$\dot{V}_{O_2}$ (l)	1.22 (0.11)	1.04 (0.17)	2.16 (0.26)	1.71* (0.21)
$\dot{V}_{CO_2}$ (l)	1.52 (0.17)	1.36 (0.26)	2.67 (0.34)	2.62 (0.45)

Values are mean (SD). $\dot{V}_E$ = Total expired ventilation, STPD; $\dot{V}_{O_2}$ = total oxygen consumption, STPD; $\dot{V}_{CO_2}$ = total carbon dioxide output, STPD; $n = 5$ for gas exchange values during 30-s WT

* Significant difference between normoxia and hypoxia

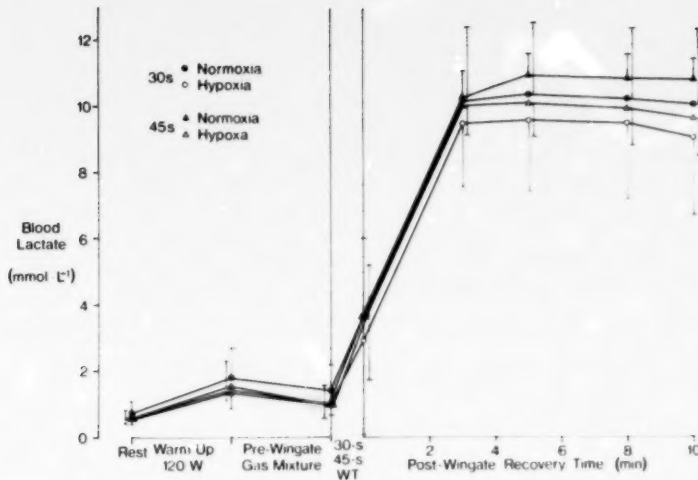


Fig. 1. Changes in blood lactate before and after the performance of either a 30-s or 45-s Wingate test (WT) during normoxic and hypoxic conditions. See the text for a description of the statistical differences among trials. ● 30 s normoxia; ○ 30 s hypoxia; ▲ 45 s normoxia; △ 45 s hypoxia

VMM 110 series ventilation module for determination of V_E . A sampling line directed dried expired gases from the mixing box to oxygen and carbon dioxide analysers (SA-3 and a CD-3 Applied Electrochemistry). Both gas analysers were calibrated before each WT trial with gases of known composition. After conversion of the analogue voltage outputs from the ventilation module and the gas analysers into digital signals (Hewlett Packard 59313 A/D Converter), V_E , V_{O_2} and V_{CO_2} were calculated using appropriate software on a microcomputer (Hewlett Packard 9825 A). For the hypoxic WT trial, the inspired O_2 fraction was sampled from the spirometer and the inspired N_2 fraction calculated immediately prior to the performance of the WT. These values were then entered into the computer programme for subsequent calculation of V_E , V_{O_2} and V_{CO_2} .

Blood samples for LA, pH and blood gases were taken immediately prior to and following the warm-up, prior to and following the WT, and at 3, 5, 8 and 10 min post-exercise. For each sample, an initial 0.5 ml blood was withdrawn from which 25 μ l of whole blood was immediately analysed for LA using an auto-analyser (YSI model 23A). An additional 1 ml blood sample was drawn anaerobically into a heparinized plastic syringe, was immediately capped, placed in an ice bath and analysed within 45 min for pH, P_{O_2} and P_{CO_2} with a pH/blood gas analyser (Corning 168) calibrated previously with precision buffers and gases of known composition. Oral temperature was taken prior to the first blood sampling interval and was used to correct all subsequent pH and P_{CO_2} values to 37°C.

Table 2. Mean power output (MPO), peak power output (PPO) and fatigue index (FI) during the 30-s and 45-s Wingate test under both normoxic and hypoxic conditions

	30-s		45-s	
	Normoxia	Hypoxia	Normoxia	Hypoxia
MPO ($W \cdot kg^{-1}$)	8.3 (0.3)	8.2 (0.4)	7.0 (0.4)	7.0 (0.3)
PPO ($W \cdot kg^{-1}$)	10.8 (1.1)	10.6 (0.6)	10.6 (0.9)	10.4 (0.8)
FI (%)	57.3 (13.0)	54.7 (6.1)	37.6 (6.8)	40.5 (7.4)

Values are mean (SD)

The muscle biopsies were analysed histochemically for fibre type (Doriguzzi et al. 1983) and biochemically for LA, CP, ATP, and glucose-6-phosphate (G-6-P) on neutralized perchloric acid extracts (Lowry and Passonneau 1972), and for glycogen as glucose units after hydrolysis in hydrochloric acid (Karlsson 1971).

A three-factor (gas $\times$ group $\times$ sampling time) repeated meas-

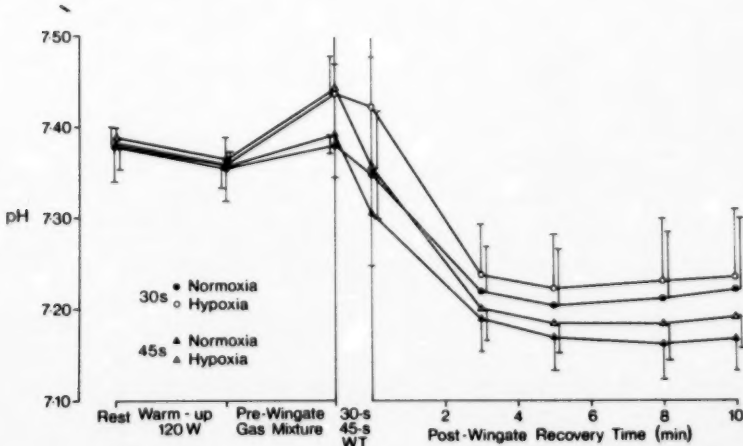


Fig. 2. Changes in blood pH before and after the performance of either a 30-s or 45-s Wingate test (WT) during normoxic and hypoxic conditions. See the text for a description of the statistical differences among trials. ● 30 s normoxia; ○ 30 s hypoxia; ▲ 45 s normoxia; △ 45 s hypoxia

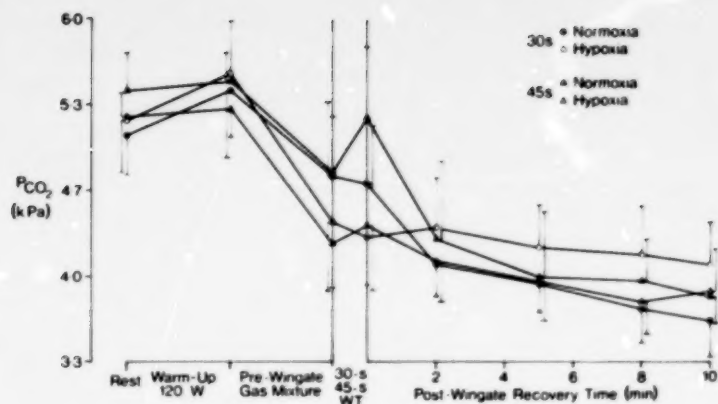


Fig. 3. Changes in blood P_{CO_2} before and after the performance of either a 30-s or 45-s Wingate test (WT) during normoxic and hypoxic conditions. See the test for a description of the statistical differences among trials. ● 30 s normoxia; ○ 30 s hypoxia; ▲ 45 s normoxia; △ 45 s hypoxia

ures analysis of variance was performed on the pre- and post-WT blood sampling data. When a significant F -ratio was obtained, a Newman-Keuls post-hoc comparison was made to clarify the differences among treatment means. Paired t -tests were calculated for the gas exchange and performance variables obtained during the normoxic and hypoxic gas conditions for each WT condition separately. For all statistical analyses, the 0.05 level of significance was used.

Results

The O_2 saturation of the blood was significantly lower for the hypoxia compared with the normoxia trial for both the 30-s and 45-s WT (Table 1). However, V_{O_2} was significantly reduced only during the 45-s hypoxic WT trial (Table 1). There were no differences for V_E or V_{CO_2} between the normoxic and hypoxic conditions for either the 30-s or 45-s WT.

Hypoxia did not impair the performance of the WT as judged by the MPO, PPO and FI during either the 30-s or 45-s tests (Table 2). As expected, the MPO and FI were lower during the 45-s than during the 30-s tests.

Blood LA increased above resting values following the warm-up and remained elevated prior to the WT for both groups during both gas conditions (Fig. 1). Following the WT, blood LA concentration increased for the duration of the recovery period. Although a similar pattern of change in the post-WT blood LA was observed for both gas conditions, there was a significant main effect of hypoxia, i.e. the values were significantly lower following the hypoxic WT.

Both WT groups showed a small but significant decrease in blood pH after the warm-up which returned to pre warm-up values prior to the performance of WT during normoxia (Fig. 2). Blood pH increased signifi-

Table 3. Muscle concentrations of adenosine triphosphate (ATP), creatine phosphate (CP), glycogen, glucose-6-phosphate (G-6-P) and lactate before and after the 30-s and 45-s Wingate test during normoxic and hypoxic conditions

	30-s				45-s			
	Normoxia		Hypoxia		Normoxia		Hypoxia	
	Before	After	Before	After	Before	After	Before	After
ATP	22.0 (2.5)	20.9 (4.1)	20.4 (1.1)	17.7 (2.5)	23.1 (3.3)	20.5 (4.1)	24.2 (2.2)	19.6 (3.9)
CP	80.6 (7.3)	50.9 (18.5)	71.4 (9.0)	26.8 (2.6)	86.4 (24.3)	44.2 (20.9)	79.8 (15.1)	41.4 (25.1)
Glycogen	275.3 (13.6)	271.7 (20.3)	309.4 (55.1)	280.4 (54.3)	345.9 (130.9)	314.5 (68.3)	359.2 (114.2)	304.4 (59.1)
G-6-P	3.5 (1.4)	11.5 (8.1)	3.6 (2.5)	19.9 (2.1)	2.9 (2.2)	16.6 (10.2)	2.7 (1.7)	16.1 (9.9)
Lactate	7.6 (7.2)	27.1 (16.9)	7.1 (7.3)	67.5* (7.9)	8.8 (6.3)	34.6 (19.1)	8.5 (3.1)	66.6* (42.0)

Values are mean (SD) expressed as $\text{mmol} \cdot \text{kg}^{-1}$ dry weight; $n = 4$ for both the 30-s and 45-s groups

* Significantly different from the value after the normoxic Wingate test

cantly while breathing the hypoxic gas mixture prior to WT; this increase carried over into the post-WT recovery period, where blood pH remained significantly higher for the hypoxic conditions compared with the normoxic trials (Fig. 2). Blood P_{CO_2} decreased significantly immediately prior to the performance of WT for both groups during normoxia and hypoxia, but this decrease was significantly greater while breathing the hypoxic gas mixture (Fig. 3). Immediately after the WT, blood P_{CO_2} remained significantly lower during the hypoxic compared with the normoxic trials. During the remaining recovery period while subjects inspired room air, there were no differences in blood P_{CO_2} between the normoxic and hypoxic conditions. Compared with the value obtained immediately following the WT, blood P_{CO_2} continued to decrease during the recovery period for the normoxic trials, whereas a significant decrease was observed only after 5 min of recovery for the hypoxic conditions (Fig. 3).

Muscle ATP and CP were significantly decreased following the 30-s and 45-s WT for both the normoxic and hypoxic conditions (Table 3). The duration of WT and hypoxia did not affect these changes. The hypoxic WT trials were not associated with a greater decrease in muscle glycogen ($P < 0.06$). Muscle G-6-P increased significantly to a similar level following the completion of each WT (Table 3). Muscle LA values increased significantly following each WT trial, but the increase was significantly greater for both groups following the hypoxic WT (Table 3).

Discussion

The normal cardiorespiratory and metabolic responses to exercise are altered by acute hypoxia to compensate for the decreased arterial O_2 content. In the present investigation, despite the respiratory alkalosis that occurred during the breathing of the hypoxic gas mixture (Figs. 2, 3), arterial O_2 saturation fell to 83% prior to the performance of WT. For the present study it is believed that the O_2 availability and tissue $\dot{V}_{\text{O}_2}$ were decreased during both the 30-s and 45-s hypoxic WT trials. This statement is supported by the lower total $\dot{V}_{\text{O}_2}$ recorded during the 45-s hypoxic WT but would appear inconsistent with the similar $\dot{V}_{\text{O}_2}$ measured during the normoxic and hypoxic 30-s WT trials (see Table 1). During the transition from rest to exercise, $\dot{V}_{\text{O}_2}$ measured at the mouth underestimates tissue $\dot{V}_{\text{O}_2}$ due to unmeasured changes in venous blood O_2 stores (Inman et al. 1987). This underestimation of tissue $\dot{V}_{\text{O}_2}$ decreases as the total exercise period increases. Consequently, the $\dot{V}_{\text{O}_2}$ measured during the 30-s trials would underestimate tissue $\dot{V}_{\text{O}_2}$ more than the values obtained during the 45-s tests. In addition, breathing an hypoxic gas mixture would decrease venous O_2 stores. As a result, the measured $\dot{V}_{\text{O}_2}$ at the mouth during the hypoxic WT trials would approximate more closely tissue $\dot{V}_{\text{O}_2}$ than the $\dot{V}_{\text{O}_2}$ recorded during the normoxic tests. Therefore, although the measured $\dot{V}_{\text{O}_2}$ was similar for the 30-s WT trials, tissue $\dot{V}_{\text{O}_2}$ likely was reduced during the hypoxic WT condition.

The results from the present investigation have shown that the performance of 30 s or 45 s of intense exercise is not impaired while breathing 10–12% O_2 . The higher muscle lactate accumulation during both hypoxic WT trials (see Table 3) implies that a greater fraction of the total ATP required comes from anaerobic glycolysis, presumably to compensate for the reduced tissue $\dot{V}_{\text{O}_2}$. Clearly, muscle LA levels measured at the end of the WT trials represent the net result of several metabolic processes (i.e. tissue release vs uptake, and production vs removal). Nevertheless, it is possible to evaluate whether the ATP equivalent for the difference in the measured $\dot{V}_{\text{O}_2}$ between the normoxic and hypoxic WT trials is commensurate with the ATP equivalent for the difference in glycogen utilization between trials. The underlying assumption for such calculations is that glycogenolysis was the only metabolic process available to compensate for the reduced tissue $\dot{V}_{\text{O}_2}$. The difference in total $\dot{V}_{\text{O}_2}$ between the 45-s normoxic and hypoxic WT trials was 0.45 l (see Table 1) or 20 mmol. Using a P:O ratio of 3, 20 mmol O_2 represents 120 mmol ATP. The change in muscle glycogen during the 45-s hypoxic WT exceeded the normoxic trial by 23.5 mmol·kg⁻¹ dry weight or 5.4 mmol·kg⁻¹ wet weight, assuming a tissue water content of 77% (Karlsson 1971). This decrease in muscle glycogen would represent an additional 16.2 mmol ATP·kg⁻¹ wet weight. If glycogen utilization in the vastus lateralis is representative of the entire active leg musculature, then to produce the O_2 equivalent of 120 mmol ATP from an increased rate of muscle glycogenolysis would require 7.4 kg active muscle mass. This calculated muscle mass is similar to other estimations of the recruited leg musculature during cycle exercise (Jansson and Kaijser 1987) or knee-extensor exercise (Andersen et al. 1985), but is less than the total leg muscle mass estimated from anthropometric measurements (Shephard et al. 1988).

As discussed previously, the difference in tissue $\dot{V}_{\text{O}_2}$ between the normoxic and hypoxic trials will be greater than the difference in $\dot{V}_{\text{O}_2}$ measured at the mouth; this would increase the calculated active muscle mass. When applied to the 30-s WT trials, similar calculations produced an active muscle mass of 2.7 kg. Although the change in muscle glycogen during the 30-s hypoxic WT exceeded the normoxic trial by 25.3 mmol·kg⁻¹ dry weight, total $\dot{V}_{\text{O}_2}$ between the conditions differed by only 0.18 l. It appears that the discrepancy between tissue $\dot{V}_{\text{O}_2}$ and the $\dot{V}_{\text{O}_2}$ measured at the mouth for this 30-s period produced the underestimation of the calculated active muscle mass.

There are at least three factors that independently or interactively could explain the greater utilization of muscle glycogen and accumulation of LA during the hypoxic WT trials. First, compared with normoxic conditions, plasma catecholamines are increased more during exercise with hypoxia (Escourrou et al. 1984). An increase in circulating epinephrine can stimulate muscle glycogenolysis (Richter et al. 1982). It could be suggested, therefore, that the different rates of muscle glycogenolysis during the normoxic and hypoxic trials

could be attributed to different levels of plasma catecholamines. However, resting levels of plasma catecholamines are not increased while breathing an hypoxic gas mixture (Rowell et al. 1986). In addition, the performance of WT under normoxic conditions is associated, by itself, with large increases in both circulating norepinephrine and epinephrine (Macdonald et al. 1983). Although the possibility cannot be excluded, it seems unlikely that differences in plasma catecholamines during the performance of the normoxic and hypoxic WT trials could account entirely for the different rates of muscle glycogenolysis.

Second, alterations in the extracellular acid-base status have been shown to influence the accumulation of muscle and blood LA during exercise (Jones et al. 1977; McLellan et al. 1988; Sutton et al. 1981). Breathing the 10–12% O₂ gas mixture induced a respiratory alkalosis prior to and throughout the performance of WT (see Figs. 2, 3). This change in the extracellular acid-base state during the hypoxic tests should be reflected intracellularly due to the rapid exchange of CO₂ across the cell membrane. During the transition from rest to exercise the rate of glycolysis is increased rapidly due to an initial intracellular alkalization (Connett 1987). This intracellular alkalization has been attributed to a Na⁺ influx and H⁺ efflux in response to muscle contraction (Connett 1987). The induced respiratory alkalosis of the hypoxic WT trials could increase this initial intracellular alkalization, either directly through changes in P_{CO₂}, or indirectly by augmenting H⁺ efflux due to a lower extracellular H⁺ concentration. The net result would be a greater initial activation of glycolysis at the onset of muscle contraction.

Third, the resistance load for each WT was calculated using 0.075 kg·kg⁻¹ body mass. Although this relative weighting for the determination of the WT load has been used by previous investigators (Inbar et al. 1983; Jacobs et al. 1982, 1983), it apparently does not represent an optimal load for the attainment of the highest peak and mean power outputs over a 30-s period (Dotan and Bar-Or 1983; Patton et al. 1985). Assuming that the rate of muscle glycogenolysis is increased to fuel the greater mean power output attained with an optimal resistance, it is possible that the predetermined WT load (i.e. 4.4 J·rev⁻¹·kg⁻¹) did not require maximal activation of muscle glycogenolysis during the normoxic WT conditions. As a result, the rate of glycogenolysis could be increased during the hypoxic trials to prevent a decrease in performance. It is interesting to note that Jacobs et al. (1982, 1983) have reported a greater decrease in muscle glycogen and a larger accumulation of muscle LA, compared with the present normoxic data, following a 30-s WT that required 4.9 J·rev⁻¹·kg⁻¹. Also, Inbar et al. (1983) have shown that the mean power output was increased during a 30-s WT (4.4 J·rev⁻¹·kg⁻¹) following the ingestion of 10 g sodium bicarbonate. These findings suggest that the induced respiratory alkalosis observed during the hypoxic WT trials may have subverted the increased rate of muscle glycogenolysis.

Despite the higher muscle LA values that were ob-

served following the hypoxic WT trials, blood LA levels were significantly reduced compared with the normoxic tests (see Fig. 1). These data suggest a decreased release and/or an increased clearance of LA following the hypoxic WT conditions. The higher concentration gradient for LA from muscle to blood, the increased extracellular pH and the possibility of elevated circulating catecholamines would all favour an augmented rather than a decreased release of LA following the hypoxic trials (Seo 1984; Stainsby et al. 1984, 1985). At the same time, the higher tissue LA concentrations should enhance the rate of LA oxidation during the recovery period (Karlsson et al. 1974). Blood flow past the tissue is also an important determinant of LA release (Eldridge et al. 1974). A decrease in the post-exercise hyperaemia following the hypoxic WT trials could explain the higher muscle but lower blood LA concentrations.

In summary, the present study has shown that the performance of 30 s or 45 s of supramaximal exercise (4.4 J·rev⁻¹·kg⁻¹) is not influenced by hypoxia (i.e. 10–12% O₂). Decreases in V_{O₂} measured at the mouth were offset by increased rates of muscle glycogenolysis and accumulation of muscle LA. It is suggested that the respiratory alkalosis, induced from the breathing of the hypoxic gas mixture, prevented the decrease in the performance of WT by increasing the rate of glycogenolysis.

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